Journal of Forensic and Legal Medicine 18 (2011) 285–287

Contents lists available at ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/locate/jflm

Case report

Suicidal plant poisoning with Colchicum autumnale

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ARTICLE INFO

Article history:
Received 13 March 2010
Received in revised form
12 February 2011
Accepted 20 April 2011
Available online 17 May 2011

Keywords:
Colchicine poisoning
Colchicum autumnale
Gastroenterocolitis
Suicidal plant poisoning
South India

ABSTRACT

Colchicum autumnale is commonly known as autumn crocus, and as *'gowri gedde'* in the southern region of Karnataka State in South India. It contains an alkaloid called colchicine, which blocks the cell division by inhibiting mitosis. We present a sporadic case of suicidal plant poisoning wherein a 24-year-old man consumed *'gowri gedde'* to end his life. Initially he presented with severe vomiting, diarrhoea and epigastric pain. He died on the third day of ingestion due to multiorgan failure. Chemical analysis of blood and viscera obtained postmortem confirmed the presence of colchicine. Colchicine poisoning is potentially life threatening because of its high toxicity and unavailability of specific antidotal treatment. It classically presents with gastroenterocolitis, and may result in multiorgan failure in fatal cases.

1. Introduction

Colchicum autumnale, a member of the family Liliaceae, is most commonly known as autumn crocus. In various parts of the world it is known by different names like naked lady, wild saffron, and meadow saffron. In the southern region of Karnataka State in South India, it is known as *'gowri gedde'*. All the parts of this perennial herb that grow from a solid bulb are toxic. Accidental ingestion of Colchicum autumnale is not uncommon as the bulb of this plant resembles wild garlic or onion. Cases of accidental colchicine poisoning due to consumption of the plant are reported.[1–5]

Colchicine is a water-soluble alkaloid extracted from Colchicine autumnale, used for the treatment of gout, familial Mediterranean fever, secondary amyloidosis, and scleroderma. Multiorgan toxicity due to accidental intravenous colchicine administration and suicidal ingestion of colchicine medications are reported.[6–10] A case is reported, where the flower of Colchicum autumnale was consumed for suicidal purpose.[11] Another two reports of self-poisoning with colchicine are published.[12,13] Also, two cases of homicidal colchicine poisoning are reported.[14] We report a sporadic fatal case of colchicine poisoning due to suicidal consumption of Colchicine autumnale.

2. Case report

A 24-year-old man consumed *'gowri gedde'* (bulb of Colchicum autumnale), to commit suicide. He was treated in a local hospital and shifted to a referral hospital for further treatment on the second day of ingestion. At the time of admission, he revealed history of 15–20 episodes of vomiting and diarrhoea since the intake of the plant poison. Stool was watery, non bloody, non foul smelling and there was no pain during defeacation. Burning sensation was present in the epigastric region. There was no history of dyspnoea, cough, palpitation, abdominal pain and fever. On examination, he was conscious and well oriented to place, time and person. His tongue was dry. Pupils were normal and reacting to light. He had tachycardia, tachypnoea and hypotension. Abdomen was soft and tender

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1752-928X/$ – see front matter
doi:10.1016/j.jflm.2011.04.008

in the epigastric region. Investigations revealed Hb – 16.4 gm; WBC – 27,000 cells/cu.mm; differential count – neutrophils 80%, eosinophils 3%, lymphocytes 15%, monocytes 2%; platelets – 1,00,000 cells/cu.mm; ESR – 2; blood urea – 102 mg/dl; serum creatinine – 4.1 mg/dl; serum electrolytes – sodium 141 mmol/L, potassium 4.2 mmol/L, chloride 101 mmol/L, bicarbonate 13.6 mmol/L. On the third day of ingestion, patient had an episode of diarrhoea with burning sensation and abdominal pain in the epigastric region. The blood pressure and heart rate were low, and the patient developed respiratory distress. He was declared dead following failure of resuscitation measures.

Medicolegal autopsy conducted on the next day revealed an oedematous and congested brain weighing 1400 gm, estimated 150 ml and 200 ml of straw coloured fluid in the right and left pleural cavities respectively, oedematous lungs with right and left lungs weighing 650 gm and 500 gm respectively, estimated 100 ml of straw coloured fluid in the peritoneal cavity, inflammed gastrointestinal mucosa, and congestion of internal organs. The heart weighed 230 gm with normal appearance of walls and valves, and patent coronaries. Stomach and proximal small intestine with their contents, one half of each kidney, a portion of the liver, and 20 ml of blood were subjected to chemical analysis. The external examination of the body was unremarkable.

Extraction of colchicine from postmortem samples was performed using ASE 300 acceletated solvent extraction system (Dionex Corp., Sunnyvale, CA, USA). The postmortem samples (stomach, small intestine, liver and kidneys) were minced into small pieces and taken separately in beakers. Each sample was mixed with around 20 gm of diatomaceous earth and loaded into 100 ml extraction cells containing cellulose filter disks (Dionex). Each sample was extracted using the conditions: temperature- 60 °C; pressure- 1500 psi; preheating period- 5 min; static extraction period- 5 min; extraction solvent- chloroform; solvent flush- 60 ml; nitrogen purge- 60 s. The extracts were collected in 250 ml sample collectors with Teflon coated rubber caps. The extracts were passed through a column containing successive layers of activated charcoal, alumina, silica gel (60–120 mesh) and anhydrous sodium sulphate. The column was eluted with chloroform. The purified extract was almost reduced to dryness by evaporation at atmospheric temperature and pressure.

Detection and identification of colchicine were done by:

2.1. Colour tests

1. To a portion of residue of the extract in a porcelain basin, a few drops of concentrated sulphuric acid were added: a yellow colour appeared.
2. To a portion of residue of the extract in a porcelain basin, two drops of concentrated sulphuric acid and a few specks of potassium nitrate crystals were added: a violet colour changing to red appeared.
3. To a portion of residue of the extract in a porcelain basin, one drop of concentrated nitric acid was added: a deep violet colour with a yellow tinge at the margin appeared.

2.2. Thin layer chromatography

The plate (20 × 10 cm) precoated with silica gel was activated in an oven at 110 °C for 1 h. The extracted tissue residue was dissolved in a small amount of ethanol and an aliquot of it was spotted on the plate. A spot of control colchicine was also applied on the same plate. Camag Twin Trough Chamber (20 × 10 cm) was saturated for 20 min with the mobile phase, methanol: strong ammonia (100: 1.5 *v*/*v*). The spotted plate was developed up to a certain distance. The developed plate was taken out and air dried. The compound was identified by comparing the Rf values of visceral extract and control sample after location of spots by using the chromogenic (spray) reagent, Van Urk reagent (one gm of p-dimethylaminobenzaldehyde dissolved in 100 ml of ethanol, to which 10 ml of hydrochloric acid was added). The developed plate showed yellow coloured spots when sprayed with Van Urk reagent.

2.3. UV spectrophotometric method

The purified extract was dissolved in ethanol and scanned in a UV spectrophotometer along with the control colchicine sample. Maximum absorption peak at 243 nm was observed for both control and visceral extract.

The diagnosis of colchicine poisoning was established by detection of the substance in the postmortem kidney, liver, and blood samples. The underlying cause of death was concluded as that due to colchicine poisoning.

3. Discussion

Colchicine blocks or suppresses cell division by inhibiting mitosis. Because of its known toxicity, its use today has been restricted to treatment of gout. Colchicine poisoning typically follows three phases: the first phase being characterised by gastrointestinal symptoms, which include burning sensation in the mouth and throat, watery diarrhoea, abdominal pain and vomiting. The second phase includes arrhythmias, liver failure, pancreatitis, ileus, adult respiratory distress syndrome, rhabdomyolysis, and hypocalcaemia that may lead to multiorgan failure and death. If the patient survives, the third phase of recovery follows during which the patient often presents with hair loss.[2,9,15] Patients with preexisting liver and renal diseases present with more severe clinical presentation.[16] In our case, gastroenterocolitis was seen during the first two days of ingestion. It was reduced on the third day, but the patient eventually developed multiorgan failure and died.

In colchicine toxicity, highly elevated blood concentrations of hepatic enzymes, creatine kinase, lactate dehydrogenase and blood urea nitrogen, as well as leukocytopenia, thrombocytopaenia and marked aplasia of bone marrow are seen.[1,17] The typical haemopoietic manifestation is initial leukocytosis followed by profound pancytopaenia, which usually begins 48–72 h after poisoning.[18] In our case, we observed thrombocytopaenia, raised blood urea and serum creatinine. But there was an increased white blood cell count in contrast to the typical picture of leukocytopenia. This probably was due to secondary infection.

At autopsy, we found inflammation of the gastrointestinal tract, oedematous lungs and brain. It is reported that centrilobular necrosis of the liver, necrosis of the proximal convoluted tubules of the kidneys, petechial haemorrhages in fatty tissues, blunt and shortened intestinal villi are seen in colchicine poisoning.[1] Histologically, there are increased numbers of mitotic figures in epithelial cells of the oesophagus and bronchioles, and in the mucosa of the large intestine and the liver.[19,20] In our case, however, postmortem samples were not preserved for histopathological examination.

As there is a widespread enterohepatic recirculation of colchicine before excretion in bile and faeces, bile is the sample of choice for toxicological analysis.[7,10,21] In our case, colchicine was detected in the postmortem kidney, liver and blood samples, by colour tests, thin layer chromatography and UV spectrophotometric methods.

4. Conclusion

Colchicum autumnale poisoning should always be considered in a case of unexplained gastroenterocolitis following ingestion of wild plants. Since there is no specific antidote available for

treatment of colchicine poisoning, it has to be treated symptomatically without delay. Our case report adds on to the literature of fatal suicidal plant poisoning with Colchicum autumnale.

Conflict of interest
None.

Funding
None.

Ethical approval
None.

References

1. Sundov Z, Nincevic Z, Definis-Gojanovic M, Glavina-Durdov M, Jukic I, Hulina N, et al. Fatal colchicine poisoning by accidental ingestion of meadow saffron – case report. *Forensic Sci Int* 2005;**149**(Suppl. 2–3):253–6.
2. Brvar M, Kozelj G, Mozina M, Bunc M. Acute poisoning with autumn crocus (Colchicum autumnale L.). *Wien Klin Wochenschr* 2004;**116**(Suppl. 5–6):205–8.
3. Gabrscek L, Lesnicar G, Krivec B, Voga G, Sibanc B, Blatnik J, et al. Accidental poisoning with autumn crocus. *J Toxicol Clin Toxicol* 2004;**42**(Suppl. 1):85–8.
4. Brncic N, Viskovic I, Peric R, Dirlic A, Vitezic D, Cuculic D. Accidental plant poisoning with Colchicum autumnale: report of two cases. *Croat Med J* 2001;**42**(Suppl. 6):673–5.
5. Hermanns-Clausen M, Schindler F, Stedtler U, Zilker T, Felgenhauer N. Poisoning by the autumn crocus plant. *MMW Fortschr Med* 2006;**148**(Suppl. 12):45–7.
6. Sussman JS, Brozena SC, Skop N, Korecka M, Shaw LM. Accidental intravenous colchicine poisoning. *Ther Drug Monit* 2004;**26**(Suppl. 6):688–92.
7. Deveaux M, Hubert N, Demarly C. Colchicine poisoning: case report of two suicides. *Forensic Sci Int* 2004;**143**(Suppl. 2–3):219–22.
8. Larrubia Marfil Y, Villamanan Bueno E, Jimenez-Caballero E, Diazaraque Marin R, Lucendo Villarin A, Fernandez-Capitan C, et al. Suicidal attempt with colchicine. *Farm Hosp* 2003;**27**(Suppl. 3):188–90.
9. Milne ST, Meek PD. Fatal colchicine overdose: report of a case and review of the literature. *Am J Emerg Med* 1998;**16**(Suppl. 6):603–8.
10. Kintz P, Jamey C, Tracqui A, Mangin P. Colchicine poisoning: report of a fatal case and presentation of an HPLC procedure for body fluid and tissue analyses. *J Anal Toxicol* 1997;**21**(Suppl. 1):70–2.
11. Danel VC, Wiart JF, Hardy GA, Vincent FH, Houdret NM. Self-poisoning with Colchicum autumnale L. flowers. *J Toxicol Clin Toxicol* 2001;**39**(Suppl. 4):409–11.
12. Ellwood MG, Robb GH. Self-poisoning with cochicine. *Postgrad Med J* 1971;**47**:129–31.
13. Allender WJ. Colchicine poisoning as a mode of suicide. *J Forensic Sci* 1982;**27**:944–7.
14. Weakley-Jones B, Gerber JE, Biggs G. Colchicine poisoning: case report of two homicides. *Am J Forensic Med Pathol* 2001;**22**(Suppl. 2):203–6.
15. Stern N, Kupferschmidt H, Meier-Abt PJ. Follow-up and therapy of acute colchicine poisoning. *Schweiz Rundsch Med Prax* 1997;**86**(Suppl. 22):952–6.
16. Brvar M, Ploj T, Kozelj G, Mozina M, Noc M, Bunc M. Case report: fatal poisoning with Colchicum autumnale. *Crit Care* 2004;**8**(Suppl. 1):R56–9.
17. Miller MA, Hung YM, Haller C, Galbo M, Levsky ME. Colchicine-related death presenting as an unknown case of multiple organ failure. *J Emerg Med* 2005;**28**(Suppl. 4):445–8.
18. Schier JG. Colchicine and podophyllin. In: Flomenbaum NE, Goldfrank LR, Hoffman RS, Howland MA, Lewin NA, Nelson LS, editors. *Goldfrank's toxicologic emergencies*. 8th ed. USA: McGraw Hill Co; 2006. p. 580–9.
19. Gilbert JD, Byard RW. Epithelial cell mitotic arrest- a useful postmortem histologic marker in cases of possible colchicine toxicity. *Forensic Sci Int* 2002;**126**(Suppl. 2):150–2.
20. Sannohe S, Makino Y, Kita T, Kuroda N, Shinozuka T. Colchicine poisoning resulting from accidental ingestion of meadow saffron (colchicum autumnale). *J Forensic Sci* 2002;**47**(Suppl. 6):1391–6.
21. Jones GR, Singer PP, Bannach B. Application of LC-MS analysis to colchicine toxicity. *J Anal Toxicol* 2000;**26**(Suppl. 6):365–9.